ASTRAGALUS CAMPESTRIS

AND

OTHER STOCK POISONING PLANTS OF BRITISH COLUMBIA

(*Figures 1 to 25*)

By E. A. BRUCE

(Animal Pathologist, Health of Animals Branch, Department of Agriculture, Agassiz, British Columbia).

DOMINION OF CANADA
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FOREWORD

It is not intended that this bulletin should cover all the plants in British Columbia that contain more or less poison, but rather to confine its scope to the more important stock poisoning plants, and to mention a few others of interest. In the main the subject matter is from the writer's actual experience covering a number of years association with the stock owners of the province: when necessary material has been taken from other sources, the excellent reports of Marsh, Clawson, and Marsh of the United States Department of Agriculture, in particular being freely drawn upon, especially in connection with larkspur and death camas.

INTRODUCTION

Considering the richness of the British Columbian flora, it is surprising to find the relatively small number of plants that cause serious trouble. Doubtless as time goes on other plants will be added to the list, and obscure losses of livestock will be traced to plants that at present may not even be suspected.

Owing to the size of the province and its varied climate, plants that are of importance in one section may be entirely unknown in another part.

In addition to the native plants one has to bear in mind that imported plants are in some districts steadily gaining ground, and may become dangerous. Examples of this can be seen in the lower Fraser Valley with foxglove, false acacia, and sneezeweed. We have already had reports implicating foxglove (*Digitalis purpurea*) as causing trouble to pigs and cattle, and the false acacia or locust tree (*Robinia pseudo-acacia*) has caused trouble with colts. The sneezeweed, yellow ox-eye, or false sunflower (*Helenium autumnale*) is known to be poisonous, but no cases have been reported as yet.

WHAT CONSTITUTES A POISONOUS PLANT?

Apart from the obvious fact that it contains some deleterious properties, there are many factors to be considered. In the first place it may not be eaten by stock although known to be dangerous, for example the common buttercups when mature are left severely alone. The amount eaten, the period of time over which it is eaten, the condition of the plant whether dry or green, the part of the plant, the kind of animal eating it, individual susceptibility, are all prime factors, and to these can be added the fact that soil, latitude, climate, and cultivation all play a part.

To argue that a plant is not toxic because an animal has been seen to eat some without harm, is no argument at all; as apart from other factors it might not be toxic for that kind of animal under any conditions. We have had such an argument advanced against the plant that forms the main heading of this bulletin, in this case because a pack-horse was seen to eat it: as a matter of fact in this instance the plant (*A. campestris*) happens to be toxic for horses, but a pack-horse native to the range might be strongly resistant, the heaviest losses being in recently imported animals, and the most susceptible subjects *par excellence* being lactating cows or ewes.

PLANTS OF SPECIAL OR UNUSUAL INTEREST

On the coast and in the moister sections of the interior the outstanding poisonous plant is the common bracken (*Pteris aquilina*), which in our experience only affects horses. Over the country as a whole the water-hemlocks or poison-parnsnips cause trouble. In the dry interior the larkspurs are of interest to cattlemen, the death camas to sheepmen, and the "timber" milk vetch (*Astragalus campestris*) to owners of horses, cattle, and sheep. The last-mentioned plant is considered the most important poisonous plant in the province, as it grows abundantly in the main ranching districts of British Columbia, and affects three different kinds of animals. It is also of interest as being a plant that until comparatively recently was considered harmless, and the disease it produces is new to science. Henry's Flora of Southern British Columbia lists twenty species of milk vetch (*Astragalus*) and seven of loco-weeds (*Oxytropis*) as occurring in the province: these two genera are of interest as containing the plants responsible for the disease known as locoism. At least one of the true locoweeds (*O. Lamberti*) occurs, but it is not abundant and the writer has yet to hear of a case of locoism occurring in the province.

Of plants of lesser importance and not mentioned in the main body of this paper, we cite: The red flowered American laurel (*Kalmia polifolia*) which grows in bogs, has produced nausea and vomiting in goats, and similar symptoms in such animals have been reported upon two occasions from eating cultivated rhododendrons. Native rhododendrons occur in British Columbia but are usually inaccessible to stock.

Attention is drawn to the fact that many cultivated plants are poisonous. We have records of three calves dying in from two to four days from eating cultivated iris, the main symptoms being salivation, swelling of the throat, irritating sores on the muzzle and lips, acute abdominal pain, diarrhœa with some blood, and death without convulsions.

Upon one occasion the eating of hydrangea caused colic, diarrhœa, and shortness of breath in a cow. The eating of a potted hydrangea (*H. Hortensia*) by a horse produced the following symptoms in about three hours: Profuse diarrhœa, the tip of the tail twisted to one side, contraction of abdominal muscles, upon attempting to lie down would only bend legs then jump into the air stiff-legged and squeal, eyes staring—recovered under treatment by veterinarian (A. J. Damman).

Upon several occasions and from widely scattered sources we have been advised by responsible stockmen that cattle feeding on the needles of freshly fallen bull pine (*P. ponderosa*) will abort. They are said not to eat the bark nor the growing needles, but will eat the freshly fallen needles greedily. We cannot affirm, nor do we deny, that this observation is correct. It is of interest, however, to state that similar reports are recorded in other parts of the world where other species of coniferous trees were concerned.

The lily (*Erythronium gigantium*), also known as the dog tooth violet and mountain adder's tongue, has been suspected as causing the death of a number of chickens and older fowls on Vancouver island. Symptoms developing in from three to five hours after they had had access to the bulbs of the plant, pieces of which could be found in the crop and intestines. Poisoned birds show a puffy crop, froth from the mouth, and drowsiness; death occurs in from two to three days.

POISONING BY YEW (*Taxaceae*)

Reports of poisoning by yew in North America are scarce, but numerous cases in various kinds of animals and man have been recorded in Europe. The only definite cases known to have occurred in British Columbia are those mentioned below, which were observed by Dr. S. Ransom some years ago.

The western yew (*Taxus brevifolia*) occurs in the province and may at some time cause trouble. It is found on the coast as far north as the Queen Charlotte islands and in the moister portions of the southeastern part of the province. Under favourable conditions it develops into a small tree with a trunk sometimes more than a foot in diameter, but usually smaller; quite frequently it is a sprawling shrub. The leaves are flat, lance-shaped, sharp-pointed, soft to the touch, and twisted on the twig in a two-ranked or feather-like arrangement; they stay on the tree for several years. The fruit is coral-red, fleshy and berry-like and contains a single seed.

In the cases reported by Dr. Ransom the incriminated plant was the ground hemlock (*Taxus canadensis*), an eastern species of yew that was being grown for ornamental purposes on a farm in the lower Fraser Valley. Four young cattle were killed and three others were ill but recovered. There was evidence that the yew bushes had recently been stripped, and this was confirmed on antopsy by finding the foliage and seeds in the stomachs of those that died. A great deal of gas was noticed in the stomachs and the lungs were greatly congested, as if the animals had died from suffocation. The extreme rapidity with which poisoning took place was shown by the fact that three of the carcases were within thirty yards of the yews, the fourth being about four hundred yards away.

THE FERN FAMILY (*Polypodiaceae*)

THE COMMON BRAKE OR BRACKEN (*Pteris aquilina*)

In sections where this fern is abundant enough to be plentiful in hay, it is responsible for heavier losses among horses than any other plant. Its dangerous nature was first drawn to public attention by Hadwen and Bruce in 1916, and the results of their investigation published in Bulletin 26, Health of Animals, Department of Agriculture, Ottawa. Their findings may be summarized in the statement that the addition of approximately six pounds of cured fern to the daily diet will kill a horse in about one month. Since that time the writer has had occasion to do further work with the plant for the following reasons: (*a*) it has been suspected by farmers on the lower mainland of causing "redwater" of cattle; (*b*) it has been said to affect sheep; (*c*) it is credited with causing a specific disease of cattle in England and a somewhat similar affection in the state of New York; (*d*) it produces the same symptoms in horses in Germany as we have in our horses, but is not reported as causing the disease seen in English cattle. We have therefore a plant that is definitely toxic for horses in two countries thousands of miles apart, credited with producing an entirely different disease of cattle in another country, that is geographically only a few miles away. The German horse cases, as with ours, are due to cured bracken in the hay, while the British cattle cases are attributed to green bracken. In so far as the plant in this province is concerned, however, the toxicity of the green or cured fern are identical, except in degree. We have produced the same symptoms in a horse fed green bracken, as occurs when the cured fern is fed; such cases, however, seldom occur in nature as we only know of two natural cases, and in these there was ample evidence to show that the horses had been compelled to eat the green fern through lack of other food. In this connection it is of interest also to note, that hay carrying sufficient bracken to kill a horse in about five weeks, can be fed to cattle all winter with apparent impunity; some cattle eat it but the majority pick out the hay and leave the fern.

In so far as our experience with green bracken is concerned we would say that neither horses, cattle nor sheep will eat it in any quantity if other food is available, in fact they seldom touch it. Even in New York state where the plant is suspected and in Great Britain where green bracken is credited with killing cattle, there is no evidence to show that the plant is eaten in any quan-

tity: in the former it is said that the disease "frequently occurs when the pastures are relatively good," and in the latter, "other food was also available in fair quantity, so that bracken was not forced upon the animals by scarcity of other food," and further that "in no case was any more than a trace of bracken found in the stomachs after death."

In the British cases we have animals dying in from twelve to seventy-two hours after showing the first symptoms; when on pasture; from a specific disease characterized by high temperatures and general hæmorrhagic lesions; said to be induced by a plant that normally they do not touch, and that can be fed experimentally (even in England) in relatively large amounts, and yet in no case is more than a trace of the plant found on autopsy. Such a combination of factors is difficult to reconcile with all the facts of the case, and certainly do not agree with our experiences in British Columbia.

THE EFFECTS OF BRACKEN ON DIFFERENT ANIMALS

Pigs.—Swine are known to root for the rhizomes which they eat without resulting harm.

Goats.—Will occasionally eat the young fronds in early spring but cease to do so when other food is plentiful.

Sheep.—Green bracken is credited with causing hæmaturia, constipation, bloody diarrhœa, convulsions, frothing at the mouth and death in such animals. Our experience would show that sheep do not eat green bracken, unless forced, and even under such conditions the symptoms mentioned do not occur (see under feeding experiments). Upon one or two occasions trouble has been reported in sheep feeding on cured or on dead standing bracken, but in these cases the symptoms could be attributed to digestive disturbances due to the food in general and not bracken in particular.

Poultry.—Upon one occasion intestinal irritation in a flock of Rhode Island Reds was attributed to the eating of finely comminuted bracken in the litter. The trouble ceased when the cured fern was removed from the straw.

Cattle.—Green bracken is never eaten in quantity or habitually in small amounts by cattle, they may take an odd bite. They can however eat a considerable amount of the cured fern along with hay without any apparent harm resulting. As with sheep a few indefinite reports of trouble have been received, which could be accounted for by the general indigestibility of the ration.

Apart from the obscure complaint in the state of New York in which green fern is suspected, and the hæmorrhagic disease attributed to the plant in Great Britain, bracken is suspected by farmers in the lower Fraser Valley of being responsible for bovine hæmaturia (redwater). Up to the present we are unable to confirm this suspicion: we have however found that the urine in such cases is on the acid side, and in the experimental feeding of bracken to cattle it was noticed that this acidity tended to increase when young bracken shoots were fed. Bracken is an acid plant and there is a possibility that the continued feeding of the cured fern in hay may be a contributory factor in redwater cases; we are not, however, prepared to admit that it is the sole or primary cause, chiefly because redwater cases do not occur in the interior sections of the province where bracken occurs, and as the fern in those sections produces identically the same symptoms in horses as does the coast fern, it is evident that the toxicity of the fern is the same.

Green bracken when fed forcibly (see under experiments) will produce gastro-intestinal irritation and even ulceration, such as could readily result in the system being flooded with bacteria from the intestinal tract, and thus result in the production of a secondary disease. To produce such a condition it is necessary to feed far more green bracken than any cattle in British Columbia have ever been known to eat naturally.

Horses.—The condition known locally as fern staggers is due to the feeding of ferny hay. Such hay will produce poisoning in horses if fed for about one month and the daily feed of cured fern is approximately six pounds. In so far as the green plant is concerned the possibility of horses being poisoned is remote, as although they may nibble an odd frond, they never eat it in quantity except under starvation conditions: only two natural cases are on record, and judging by the one case in which the green plant was fed experimentally the amount necessary to produce toxic symptoms is far in excess of that required by the cured fern.

SYMPTOMS IN HORSES

The earliest is probably a yellowish tint of the mucous membranes of the eye. Animals that normally lie down at night may cease to do so. The breathing may be a little hard and the animal appear to drag the legs. Usually the first symptom noticed is uncertain gait. There is an appearance of intoxication. If the head is raised the symptoms are more pronounced and the animal may fall. The pupils of the eye may be dilated. The appetite is usually good; some cases have been reported where there was some difficulty in swallowing but this has not been noticed by the writer. By pushing on the side of an animal it may be possible to rock it back and forth. There is a tendency to constipation. The pulse is usually weak and in the later stages quickened. The temperature is never elevated but may be subnormal. The head is usually carried low. In one of the two natural cases that we have seen due to green bracken, the animal when lying down was noticed to nod its head in a peculiar manner for several minutes. Unsteadiness of gait may be in evidence for several days but others go to the ground quickly. Once down it may be possible to raise the animal, but it is seldom possible to do so after it goes down the second time, such cases are usually doomed. When down the animal may still eat; usually there is a lot of struggling and some cases show tetanic spasms. Twitching of the muscles, especially of the face are common, and nervousness is well marked. The animal batters itself about badly and gets progressively weaker until death occurs.

TREATMENT

Veterinary aid should be secured if possible. Remove all ferny hay and bedding. Give an aloetic ball or a quart of raw linseed oil, good clean hay, warm bran mashes and roots or other green feed if available. Keep the animal warm, and place in slings if necessary. Care is needed in giving medicines as rough handling will aggravate the nervous symptoms. The animal must be kept as quiet as possible. In some cases cold packs to the head are useful. Any line of treatment should be started early, as once the animal is down its chances of recovery are lessened.

FEEDING EXPERIMENTS

The feeding of cured bracken and subsequent poisoning of horses by Hadwen and Bruce, can be found in Bulletin 26, Health of Animals, Department of Agriculture, Ottawa (1917).

The following feeding tests were made to determine the effect of the plant on cattle and sheep; the feeding of green bracken to a horse being undertaken largely as a check on the toxicity of the bracken fed to calves.

Sheep.—A ram absolutely refused to touch green bracken although no other food was offered for two days.

Ewe.—This animal from August 15 to August 28 was offered on alternate days no other food but green bracken; on such days the animal refused to eat. From August 28 until October 5 it was literally starved into eating green fern as no other food was offered. The animal lost a lot of flesh (which was subsequently regained on pasture) but did not show any symptoms of poisoning.

Cattle.—Heifer 3 years. Fed 128 pounds of green bracken from September 2 to the end of October, with negative results. Heifer 3½ years. Fed one kilo per day of cured bracken from October 25 to April 27, approximately 408 pounds, and from that date until July 13 one kilo per day of the green fern, approximately 170 pounds. This experiment as well as the preceding were negative in so far as the production of redwater was concerned; beyond a slight increase in urinary acidity towards spring, which was transitory and probably due in part to timothy hay, no symptoms of poisoning were noted.

With a view to reproducing, if possible, the results obtained by Stockman in England, excessive amounts of green bracken was fed as follows:—

Calf 1, age 5½ months. With the exception of a small amount of feed that this animal could pick up when allowed to exercise for an hour or so each day, its diet was exclusively green bracken from August 4 (1925) to September 12, i.e., forty days.

The amount consumed was 495 pounds, which is 235 pounds more than was fed to the heaviest fed calf in England, which was also older, being 8 months of age. During this time no abnormal elevation of temperature occurred, and except for marked loss of flesh no signs of illness were noted. On September 14 the animal was killed and autopsy showed the following: The fourth stomach showed some congested areas; the small intestines in a catarrhal condition, the mucosa thickened and corrugated and in some places inflammed, no ulceration was noted nor was any blood exuding from the congested mucosa. Inflammation very well marked around the ileocæcal valve but no bleeding; a few spots suggesting minute ulcerations were noted in this area but were evidently due to whip-worms, which were present. The liver appeared normal but the kidneys showed some congestion. The mesenteric glands were enlarged, watery on section, and invaded by *micrococcus tetragenous.* A few petechial spots were present in the rectum. The large intestines were not inflammed, and the lungs and other organs appeared normal.

Calf 2, age 3½ months. Fed in the same manner as Calf 1. From October 5 (1925) to November 4, thirty-one days, this animal ate 264 pounds of green bracken (a calf of the same age was fed 112 pounds in England). During the time of feeding this animal showed no ill effects beyond loss of flesh, nor did it show any marked variation in temperature. When bracken feeding stopped it was kept on pasture. Fourteen days later (November 18) the calf was off feed and carried a temperature that ranged from 105·6 to 106·2. On the 19th the temperature ranged from 103 to 103·6. On the 20th the morning temperature was 102·2, at noon 101, and at 2 p.m. was 100·8 at which time the animal was killed. Upon autopsy the cause of the sudden illness was clearly shown to be due to rupture of an ulcer in the small intestine, which had subsequently partially healed, the omentum being adherent to the affected bowel.

Apart from this evidence of a mild peritonitis, the outstanding lesions in this calf consisted of a number of small ulcers, rarely larger than one-quarter of an inch in diameter, which were confined to Peyer's patches: these were filled with necrotic tissue and carried a mixed bacterial flora, and in many instances were deep enough to involve the muscular coat. The adjacent lymphatics were slightly swollen and contaminated by bacteria. The stomach appeared normal, and apart from the ulcerated condition the intestine showed little beyond a mild catarrhal condition. The kidneys showed a mild nephritis and the liver congestion. Other organs appeared normal.

The following experiment was conducted with a view to showing that green bracken gathered at the same time of the year and incapable of producing the so-called bracken poisoning in calves, would produce toxic symptoms in a horse similar to that seen in horses poisoned by cured fern.

No data as to the amount of green fern necessary to affect a horse was available, as the only cases on record were the two natural cases reported by Hadwen and Bruce.

A suitable animal was not obtained until October 24 (1925); feeding started on that date and was continued until November 29, a total of thirty-seven days, very little, however, being eaten on the thirty-seventh day. The horse used was seven years old, 1,250 pounds, and sound except for slight pulmonary emphysema. Green bracken was fed with the exception that on November 9, 12 and 13, some, and from the 26th on, only brown standing bracken was fed. During the test the animal consumed 1,038 pounds of green bracken and 413 pounds of dead bracken, which is not to be confused with cured fern as found in hay, of which approximately 200 pounds will kill a horse. On the thirty-sixth day of feeding the animal showed slight inco-ordination, temperature 99·6. On the afternoon of the thirty-seventh day it fell and was unable to rise, temperature 98·2. The following morning the temperature was 97·3, the animal weak, intoxicated and unable to rise; by afternoon its temperature had dropped to 96·2, pulse 84 and very weak —a typical case of bracken poisoning, would still eat hay if offered, nervous, lips twitching, conjunctiva yellowish, and if rolled onto sternum always kept the head hanging down.

Comparing this case with those due to cured fern, it is to be noted that the period of feeding to produce symptoms was about same; the amount of bracken fed was far in excess of that necessary to affect horses with the cured plant; the symptoms and lesions correspond allowing for differences we have noted in different individuals. Apart from the large amount of green fern required, the one point that stands out is that the animal stayed on its feet for a shorter time and got weaker faster than we would have expected.

Autopsy.—The animal was killed the afternoon of the thirty-eight day and autopsy showed: inflammation of the pyloric end of the stomach, bots present and alive, no ulceration; the small intestines corrugated, inflammed in places and covered with a greenish-yellow mucous, inflammation most marked towards lower part of the ileum. No inflammation noted in large intestines. The bladder showed a few petechial spots and the urine contained albumin. The liver friable, dark, and on section showed degeneration. The kidneys showed nephritis. Brain and cord congested. No evidence of bacterial infection. Other organs normal with exception of emphysema of the lung, which was present before feeding started.

COMMENT ON FEEDING GREEN BRACKEN TO CALVES

The green bracken gathered at the same time of the year as that which was toxic for a horse, caused in two calves some gastro-intestinal irritation but did not cause an intoxication such as is common to horses poisoned by either the green or cured fern. Notwithstanding the fact that we fed greater amounts of green bracken to these calves than was fed by Stockman in England to any of his calves, we were unable to produce the disease designated as bracken poisoning of cattle in the British Isles. We can agree with Stockman only to the extent that continued heavy feeding of green bracken will produce gastro-intestinal lesions. These lesions are such as to readily allow bacteria from the intestinal tract to enter the general system. It is evident that negative results follow the feeding of green bracken in moderate quantity to cattle, even if fed for some time. Apart from the calf feeding we have negative feeding tests in England of 56 pounds in six days and 60 pounds in seven days; 40 pounds in a few days in New York; and in British Columbia 128 pounds in sixty days, 170 pounds in seventy-seven days, and 408 pounds of the cured fern in one hundred and eighty-four days: in all cases fed to cattle old enough to be designated as heifers or cows.

Neither in England, in the state of New York, nor in British Columbia is there evidence to show that green bracken is eaten in any quantity by cattle when on pasture if other food is available.

SUMMARY

(1) Cured bracken as found in hay causes heavy losses in British Columbian horses.
(2) Only two natural cases due to green bracken have been recorded in horses, and these were induced by starvation.
(3) The green or cured plant produces similar symptoms in horses.
(4) The symptoms indicate an intoxication: unsteadiness in gait, loss of balance, reddish-yellow conjunctivae, dilated pupils, good appetite, constipation, nervousness, weakness, muscular twitching, spasms and death.
(5) The amount required to produce poisoning is approximately six pounds of cured fern per day for one month.
(6) Cured bracken is and may be eaten by cattle in moderate amounts without harm.
(7) Experimentally it is possible to produce gastro-intestinal lesions in calves by feeding green bracken heavily, and the lesions are such as could give rise to secondary infection.
(8) At the present time we have no evidence to show that bracken will produce hæmaturia (redwater) in cattle.
(9) In our opinion sheep are not poisoned by bracken.
(10) Our experience would show that the green plant is never eaten in quantity or habitually, if other food is available, by either horses, cattle or sheep in British Columbia.
(11) Our findings are not in accord with those of Stockman in so far as green bracken poisoning of cattle are concerned.

EQUISETACEAE *(Horsetail Family)* (Fig. 1)

Several species of Equisetum occur in British Columbia, but the only one that causes much trouble is the common field horsetail (*E. arvense*). This plant is often abundant in hay, and has caused sickness and death of horses in various parts of the province. In so far as our records go, only horses are affected.

For all practical purposes the symptoms and treatment are the same as those given for bracken poisoning of horses. There is, however, usually more unthriftiness and less tendency to constipation in the early stages than in bracken poisoning, and the animal as a rule stays on its feet longer, the loss of the sense of equilibrium is not so marked, after falling it can get up oftener, and the conjunctiva is not as reddish-yellow. We are not able to mention any one outstanding symptom to differentiate the two diseases, but where bracken does not occur plentifully in the hay horsetail should be looked for; occasionally both are present at the same time. It usually takes some weeks of feeding to produce poisoning. Some cases are quite acute but generally not nearly so acute as bracken cases. At times chronic cases occur which hang on for weeks, a condition that does not exist with bracken poisoning, so far as the writer is aware. Such cases are benefited by a course of nerve tonics, such as nux vomica.

Equisetum poisoning is by no means as common as bracken, but on the other hand is not uncommon. The green plant does not appear to cause trouble.

It is perhaps of interest to mention a very early record of another type of equisetum poisoning that was drawn to my attention by Mr. J. W. Eastham. About the year 1860 John Keast Lord spent four years in British Columbia; he was a veterinarian by training and was engaged as naturalist to the Boundary Commission then marking the boundary between British Columbia and the state of Washington. His reference to equisetum poisoning occurs in his book "At Home in the Wilderness" 2nd ed., 1867 . . . "the gold discoveries on the

Fraser River "Bars" attracted a vast concourse of miners, and consequently mule trains, for the purpose of supplying the diggers' necessities. When the cold weather came on the mule trains were, nearly every one, driven down to the Sumass and Chilukweyuk prairies, in order to winter the animals . . . snow rapidly covered up the grass far too deeply for the mules to dig it away with their feet, in order to reach the buried herbage. No dry fodder had been provided to meet this contingency, so, in the absence of all other kinds of foliage, the hungry mules began to devour the large patches of "equisetum," or horse-tail rush, which covered many acres of ground under the trees by the river side, and around the swampy edges of the bush; being in a great measure protected by the trees, and growing often to a height of six feet, it was easily comeatable above the snow. The effect of the plant was perfectly astounding. As soon as the mules began to eat it they were seized with a disease precisely resembling Asiatic cholera; the most violent purging came on, accompanied by cramp, rigors, utter prostration, and speedy death. More than five hundred mules died on these prairies in less than a month. . . . I have often seen the mules eat this horse-tail rush during the summer, when mixed with other food, and then no ill effects accrued from it".

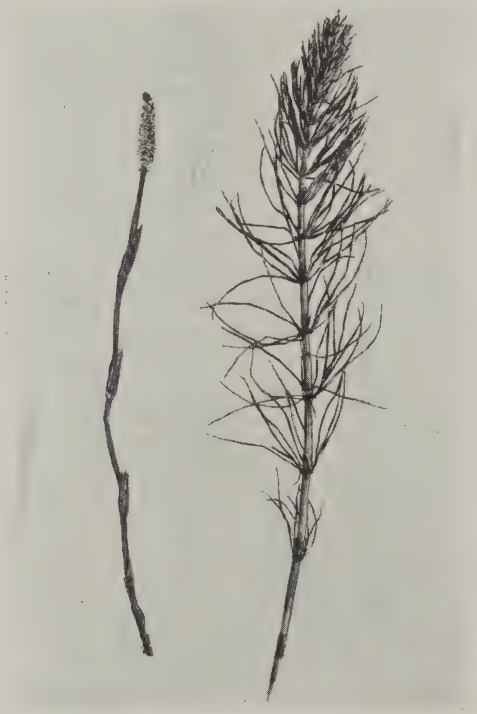

FIG. 1.—The common field horsetail *(Equisetum arvense)*. The fertile form on the left, which soon dies down, and the sterile stage which is bushy and is found in hay.

(Photo by Glendenning)

Judging by the location and the size of the plant, together with the fact that it was strong enough to show through the snow, the equisetum referred to by Lord is the Scouring Rush (*E.hyemale var. robustum*).

While it is possible that other varieties of equisetum may cause trouble in British Columbia, the one that needs to be especially considered is the common field horse-tail (*E. arvense*), which is fond of damp sandy soil, and is from four to twenty inches in height.

THE LILY FAMILY *(Liliaceae)*

DEATH CAMAS *(Zygadenus* sp.) (Fig. 2)

At least three species of *Zygadenus* occur in British Columbia, but the commonest is the death camas, *Z. venenosus*, which has a wide distribution. All species are poisonous, and all have green grass-like leaves, which, at the early stages of growth, are not readily distinguished from the grasses, but are somewhat thicker. The plants are erect and grow from a bulb, which in most cases is too firmly fixed in the ground to be pulled up by grazing animals. The flowers are greenish-white or greenish-yellow and grow towards the top of the single stem; in the interior they are in bloom in June and somewhat earlier on the coast. While some of the species reach a greater height, the death camas (*Z. venenosus*) does not often exceed a foot in height, but varies from about eight inches to a little over two feet.

FIG. 2.—The death camas *(Zygadenus venenosus)*. Plant on the left half the size of the scale, that on the right full size compared with scale.
(Photo by Glendenning—from painting by Young.)

ANIMALS POISONED

Horses and cattle may be poisoned, but the majority of cases are in sheep and it is only in such animals that cases have occurred in British Columbia, in so far as we know definitely. Human beings are occasionally poisoned through mistaking the bulb for that of the edible camas, species of Calochortus and Camassia, or for the wild onion.

PARTS OF THE PLANT POISONOUS AND AMOUNT NECESSARY

According to Marsh and Clawson the toxic dose for sheep (to which animals fatal cases are almost entirely confined), varies from 1·6 pounds to 5·6 pounds per hundredweight of animal, in animals feeding naturally; the wide range in amount being accounted for by the time taken in feeding. In forced feeding or drenching experiments the toxic dose was about half a pound, and it is thought that this amount more closely represents the toxic dose in a hungry sheep feeding naturally where feed was short and the death camas abundant. The plants are poisonous throughout their whole growth. The tops and bulb being equally poisonous, while the seeds are much more toxic than other parts, the toxic dose with them being only a little more than one-tenth of a pound. Cases of poisoning are more likely to occur before the plant is fully matured, because at that time other forage is scanty.

SYMPTOMS OF DEATH CAMAS POISONING

In acute cases frothing at the mouth is nearly always seen, often accompanied by grinding of the teeth. Nausea is common and quite commonly vomiting occurs. At this stage the breathing is quickened but later it slows down and the animal may lay for hours breathing slowly. The period of quiet may be broken at intervals by spells of rapid breathing accompanied by panting and struggles lasting for a few minutes. Trembling and sensitiveness to sudden noises or movements may be noticed. In severe cases muscular weakness is well marked. In cases where the animal is strong enough to stand, the gait is usually stiff, all the legs being affected but more noticably the hind. In severe cases the animal will lie for hours or even days with laboured breathing, in an unconscious condition, ending in death without any spasms.

TREATMENT

There are no remedies known that are of practical value. Sheepmen often bleed affected sheep but this is of no material benefit. The drug most frequently recommended is potassium permanganate in 5 to 10 grain doses, but this treatment when tested experimentally by Marsh was found to be worthless: better results were obtained by repeated doses of tannic acid or bicarbonate of soda, and it is possible that potassium permanganate might be of value if used in the same manner. Any of these drugs are given with sufficient water to make a drench, but to be of any value must be given frequently at intervals of not more than half an hour apart. The dose of tannic acid for sheep being half a teaspoonful, and of sodium bicarbonate (baking powder) a teaspoonful.

The poisoned animals should not be disturbed any more than is absolutely necessary.

Cases of poisoning are most likely to occur before the death camas is in flower, not because it is more dangerous at this stage but because other forage is liable to be scarce. Sheepmen should learn to know the plant at this stage so that they can avoid it. If sheep have to be driven across a patch where the plant is known to be abundant, they should be well fed before starting, as it is the hungry animal that is most likely to be poisoned.

THE FALSE HELLEBORE *(Veratrum viride)*

The false hellebore or Indian poke, is the largest and handsomest greenish-flowered plant growing in the mountains; it grows from 2 to 7 feet in height, and has large oval, strongly parallel veined, pointed leaves, often four inches or more in width; the inflorescence composed of yellowish and later greenish coloured flowers may be two feet in length.

The whole plant is known to be more or less poisonous but is seldom touched by stock.

The only definite record of its affecting stock in this province that has come to our notice, is that a number of sheep after eating the *blossoms,* staggered and became temporarily blind, but recovered.

Definite data as to under what conditions the plant is poisonous, are lacking: sheep are said to sometimes eat the leaves without harm, and with impunity after they have been frozen.

THE BUCKWHEAT FAMILY *(Polygonaceae)*

BUCKWHEAT POISONING

That the cultivated buckwheat (*Fagopyrum esculentum*) can cause poisoning of stock under certain conditions is not as well known as it might be, the subject being one that is not often mentioned in bulletins of this nature. To produce poisoning, technically known as fagopyrismus, three factors are essential: they are the feeding of buckwheat, an uncoloured skin, and sunshine; if any one of the three are lacking poisoning does not occur.

The plant is said to be most toxic when in flower, and to have caused fatal cases in the ox, sheep, pig, and rabbit. Animals with pigmented skins do not suffer, and white animals show no ill effects if kept out of direct sunshine. Even when the feeding of buckwheat has been discontinued for ten days, or for two to three weeks, the eruption will develop if susceptible animals are exposed to the sun. The more buckwheat an animal has eaten and the hotter the sun, the greater will be the reaction.

SYMPTOMS

In mild cases there is reddening and slight inflammatory swelling of the skin accompanied by intolerable itching. The region of the head and ears being the most commonly affected. In severer cases there may be some disturbance of breathing, digestive trouble, and symptoms of brain affection. Death may occur in twelve hours.

The following interesting outbreak in pigs occurred during August, some years ago, in the lower Fraser Valley:—

A number of Yorkshire pigs had been running on a pasture of rye grass and other green feed for ten weeks; they were then moved to a similar pasture which contained, in addition, buckwheat in flower. They were on this latter pasture only 24 hours when symptoms developed. The ages ranged from three months to aged brood sows, and all exhibited the same symptoms, the younger pigs being affected first.

The symptoms noted, in addition to a reddened inflammed skin were those of a brain affection. They would try to jump out of the enclosure; would jump into the air with all four feet off the ground at the same time; shake their heads and squeal, then poke their heads against the pen or under a log, where they would remain quietly for three or four minutes and then begin to act as before. At times the hind legs were partially paralyzed. (Dr. G. Howell.)

The feeding of buckwheat was suspected as being the cause of the trouble by those in charge, but the true nature of the disease was not understood, because on the following day the pigs having eaten some food and apparently much better, were turned out onto a ploughed field in the hot sun without shelter of any kind. The result being that in a few hours they were showing the same symptoms as before, but not quite so well marked. All subsequently recovered under proper treatment.

TREATMENT

Affected animals should be moved at once to a shady situation. As a rule recovery takes place in a few days if this is done, but the severer cases should receive treatment to reduce the inflammation of the skin, especially those showing brain or general symptoms.

SOMEWHAT SIMILAR CONDITIONS

In various parts of the world the following plants have been reported as causing skin irritation in white skinned animals: Red clover, bur clover, alsike clover, St. John's wort, and alfalfa. In the dry interior of British Columbia alfalfa has been known to cause inflammation, followed by scab formation on the white noses and fetlocks of horses; white pigs are also said to suffer. Several of the knotweeds or smartweeds (*Polygonum sp.*) are also considered harmful, but our feeding experiments have not confirmed this. That there is some plant in the Fraser Valley capable of causing similar trouble, is evident, as at one time we saw three out of a lot of nineteen Holstein heifers badly affected. These animals had been running in the woods and the only plants found that could be suspected were knotweeds. Only the white parts of the skin were affected, the black parts being perfectly normal while the white was badly inflammed. The two worst cases had a temperature of 104°, and showed a reddening of the mucous membrane of the eye. The worst case was treated with copious applications of carron oil, and made a slow recovery. The other two were given no treatment but kept under observation. The affected skin all peeled off, and in one animal a patch ten inches square in the loin region was so badly damaged that no hair grew on it again. To try and find the cause different species of knotweeds were fed as follows: Two pigs were fed ten days in the shade and then were placed in the sun: in addition to shorts one got 70 pounds of lady's thumb. (*P. persicaria*), the other 70 pounds of red clover (*T. pratense*).

Three pigs fed nineteen days in open air, one received 135 pounds lady's thumb, the second 135 pounds red clover, the third 90 pounds of water-pepper (*P. hydropiper*).

The results were negative beyond a slight reddening and scabiness of the ears which was most marked in the lady's thumb pig.

A yearling Holstein bull was fed eighteen days in the open on polygonums exclusively. It received 140 pounds of lady's thumb, 140 pounds of water-pepper, 40 pounds of *P. aviculare*, 30 pounds of *P. acre*, and 10 pounds of *P. hydropiperoides*; total 360 pounds. Results were negative, no ill effects being noted.

Both with the bull and the pigs the plant that was eaten most readily was the lady's thumb (*P. persicaria*).

THE BUTTERCUP FAMILY *(Ranunculaceae)*

THE LARKSPURS *(Delphinium* sp.) (Figs. 3 & 4)

Although a number of the Buttercup Family are more or less poisonous the only plants with which stockowners in the province have had serious trouble are the larkspurs. Seven different species grew in British Columbia, and are widely distributed, the majority being found in the interior of the province. They can be divided into two groups; the tall leafy species which may reach six feet in height, such as *Delphinium glaucum* and *D. Brownii*, and the low larkspurs (*D. Menziesii, D. bicolor*) which rarely exceed a foot in height and which have but few leaves except towards the base. The low larkspurs are usually dried up by July but the taller forms persist until later in the season, and may not entirely disappear until broken down by the winter snows. Early in the season before the flowers appear the leaves of the tall larkspurs may be mistaken for those of the wild pink geranium which is quite harmless; they can be distinguished with certainty by examining the base of the leaf-stem, which in geranium will show two small leaves (stipules) which are absent in larkspur. At a later stage of growth the tall larkspurs may be confused with aconite, which itself is poisonous but does not cause trouble because of its limited distribution and the fact that it is seldom eaten by stock. Both plants have almost

identically shaped leaves and grow to the same height; both have blue flowers but as the shade of colour varies from deep blue to white in aconite, colour is not a good distinguishing feature. The shape of the flowers, however, is quite different: larkspurs are characterized by the back of the flower carrying a well marked spur, as the common name of the plant would indicate—aconite, commonly known as monkshood, carries in the same position, not a spur, but a well marked hood or cowl.

FIG. 3.—Low larkspur *(Delphinum bicolor).*
(Photo by Glendenning)

PARTS OF LARKSPURS POISONOUS

Experiments conducted by Marsh, Clawson and Marsh of the United States Department of Agriculture prove conclusively that all parts of the plant are poisonous, and that the seeds are especially so. In almost all cases poisoning is due to eating the leaves and flowers. Most of the cases occur in the spring or early summer.

ANIMALS AFFECTED AND AMOUNT NECESSARY TO CAUSE POISONING

Horses can be affected experimentally, but under natural range conditions they never eat enough of the plants to become poisoned. Sheep can be grazed upon larkspur without harm. Cattle, however, are so frequently poisoned as to cause American writers to state that probably no other plant, with the exception of the loco-weed, has caused such heavy losses to stockmen in the western United States. In British Columbia it causes trouble from time to time in the interior of the province. According to Marsh and Clawson the amount necessary to produce poisoning is approximately 3 per cent of the animal's weight.

but it may eat two or three times as much without showing signs of intoxication. In the general average of cases cattle weighing 1,000 pounds will be poisoned if they eat as much as 60 pounds in one day: the quantity varies, however, within wide limits, in one case being as low as 30 pounds, and at the other extreme as high as 93.3 pounds.

FIG. 4.—Low larkspur *(Delphinium Menziesii).*
(Photo by Glendenning)

SYMPTOMS OF LARKSPUR POISONING

The characteristic symptoms are nausea, weakness, excessive salivation, twitching of the muscles, and convulsive movements. On the range usually the first indication of trouble is the sudden falling of an animal, which struggles violently to rise but is unable to do so immediately. Later it may get up and walk off as if nothing had happened, but more frequently it falls again, and is almost certain to do so if hurried. Falling may be repeated several times. Before falling the animal may be noticed to be somewhat stiff in its gait and the hind legs widely spread to brace itself; it may be noticed to move backwards, the back arched, the head low, and show some trembling. When down in light cases the head is held up, but in severe cases the animal lies upon its side with the feet extended more or less rigidly, sometimes moving the head up and down. Bloating and increased salivation may be noticed but neither are constant symptoms. Nausea is shown by the belching of gas at frequent intervals, the animal moving the head back and forth and sometimes shaking it from side to side. Vomiting sometimes occurs and such cases usually die. The breathing is quickened. Twitching of the muscles may occur around the face, shoulders,

flanks or hips, and sometimes muscular trembling may be noticed all over the body. Some cases moan and show evidence of abdominal pain. Constipation is commonly present. Recovery from larkspur poisoning is usually rapid, as also is death. Dead animals bloat rapidly. The same symptoms are produced by both the tall and low larkspurs.

TREATMENT AND PREVENTION

The larkspurs are not violently toxic plants and can be eaten in considerable quantities by cattle without harm, it is however always dangerous to allow such animals to graze on them freely. Hungry animals being driven over such areas should be especially watched. Most of the cases occur in spring and early summer at which times heavily infested areas can be used for sheep, as they are never affected, or for horses which are not known to suffer under range conditions. The presence of a few larkspurs need cause no concern to the cattleman, but large quantities are dangerous.

In cases of poisoning the affected animal should be turned so that its head is higher than the body; no attempts should be made to get it on its feet but it should be allowed to lie quietly. If the animal gets up it should not be hurried. Bleeding serves no useful purpose and is not recommended. If bloating occurs the paunch should be punctured with a trocar. As the plant causes paralysis of the organs connected with breathing and constipation is always present, the logical treatment is to correct this. March and his associates recommend the following remedy which has to be given under the skin with a hypodermic syringe:—

Physostigmin salicylate	1 grain.
Pilocarpin hydrochlorid	2 grains.
Strychnin sulphate	½ grain.

This formula is for an animal weighing 500 or 600 pounds. For an animal 1,000 pounds or more twice the amount should be used. The physostigmin salicylate and pilocarpin hydrochlorid is sold in tablets, each tablet containing one-half grain of the physostigmin and one grain of the pilocarpin. The strychnin sulphate can be bought in half-grain tablets. With such tablets the dose for the 500-pound animal would be one strychnin tablet and two of the physostigmin-pilocarpin tablets; for the larger animals two of the strychnin and four of the physostigmin-pilocarpin tablets. These dissolve readily in water, two ounces of which should be used for a dose. It can be mixed up ready for use before going on range if desired. The prompt use of this remedy will result in saving most cases. Where no such remedy is available, the main things to remember are to keep the animal's head uphill, and to let it lie quietly.

THE PEA FAMILY *(Leguminosae)*

ASTRAGALUS CAMPESTRIS GRAY (The "timber" milk vetch)

A disease is caused by the ingestion of the "timber" milk vetch (*Astragalus campestris* Gray) which differs entirely from that produced by any of the other known toxic species of Astragalus, or the closely related genus Oxytropis, in which genera the plants responsible for locoism are found. This disease does not resemble locoism at all, but in some respects suggests lathyrism.

The affection is characterized by inco-ordination, difficult respiration accompanied by wheezing, whistling or roaring, and loss of voice in cattle and sheep, in which animals it is commonest in lactating females. Horses are also affected, the most prominent symptom being roaring of an expiratory type. Although indigenous to British Columbia the disease does not appear to have a common name: it is variously referred to as,—timber trouble, timber grass poisoning, jack pine fever, husky, Kamloops cattle disease, knocking disease, timber

paralysis, and roaring disease, these names applying to cattle and sheep. In horses it is known as,—mountain fever, roaring disease, Clinton horse disease, and timber paralysis. We prefer the terms timber paralysis and roaring disease.

AFFECTED DISTRICTS AND CLIMATIC CONDITIONS

In British Columbia there exist a series of longitudinal belts of climate which, when contrasted with each other, show great differences, especially in precipitation. From west to east, these belts are named as follows: Coastal, Dry, Interior Wet, and Rocky Mountain belts.

All known cases of the disease have occurred in the Dry belt, with the exception of cases in horses and cattle in the vicinity of Windermere lake in the Rocky Mountain belt, where climatic and floral conditions simulate those of the dry interior. A large tract of country is involved as the disease is found near Tatlayoko lake, in the Chilcotin, lac la Hache, Clinton, Ashcroft, North Thompson, Kamloops, Nicola, Okanagan, and Similkameen districts, as well as in the Windermere Valley. It extends from north of latitude 52 degrees to the international boundary in the vicinity of Chopaka, and if information we have is correct also occurs in Okanagan and Ferry counties in the state of Washington. In British Columbia it is roughly estimated that nine or ten thousand miles of range is affected. The disease develops when stock are ranging in the Arid Transition zone, to which *Astragalus campestris* belongs. This zone lies between an altitude of approximately 2,000 feet and 4,200 feet, but may run a little further up or down. It is divided into two subdivisions, the bunch grass prairies that lie immediately above the sage brush (*Artemesia tridentata*), and the timbered area which consists mainly of Douglas fir. In some areas, due to repeated fires, the Douglas fir is replaced by the lodgepole pine (black or jack pine). Scattered throughout the timber are open grassy glades of varying extent. At the upper limit of the zone the Engelmann spruce appears.

The total precipitation over this zone can seldom be more than 15 inches a year, and in some sections is considerably less, e.g., Clinton, altitude 3,040 feet, has a total of 5·7 inches.

The northern slopes of the hills being somewhat moister, produce a more luxuriant vegetation than those facing south, and although affording better pasturage are more dangerous in so far as the disease in question is concerned. It is generally conceded that most cases occur in those years in which the summer is unusually dry.

DESCRIPTION OF "ASTRAGALUS CAMPESTRIS" GRAY

The "timber" milk vetch is one of the most conspicuous plants on the affected range, growing both in and out of the timber. It is from 8 to 16 inches high and grows in rounded tufts, the taller specimens being found in the moister locations. It is a pretty, fragile vetch, with fine grass-like stems, small narrow leaflets, and little, scanty, pea-like pinkish-mauve blossoms, which grow comparatively far apart in long slender racemes. The colour of the flowers varies considerably, some specimens being almost white and other bluish; the majority, however, are pinkish-mauve. It is difficult to describe a plant adequately in simple language, but it is hoped that the foregoing together with the illustration (fig. 5) will suffice. The botanical description follows:—

Slightly pubescent, tufted, 2-4 dm. high, stipules membranaceous below, less so above; leaflets 7-13, distant, 2 cm. long, narrowly oblong to nearly linear; peduncles long and slender; flowers few to many, whitish to mauve, tinged with purple, 8-19 mm. long; calyx-teeth one-third the length of the tube, some black hairs among the pubescence; pod linear-oblong, not much flattened, 2-2·5 cm. long, lightly pubescent or glabrous, finally deflexed. Very common, Lytton eastward. (Henry's Flora of Southern British Columbia.)

SEASONAL PREVALENCE

Cases of poisoning correspond with the period in which the plant is apparently suitable for food. On the lower levels this may be as early as the latter part of May, but the majority of cases occur during the last half of June and through July, further trouble occurring until towards the latter part of August, by which time the plant has finished seeding and dried up. After drying up the plant is often eaten and apparently without harm. It is eaten to a certain extent at all times, but heavy consumption is closely related to shortage of other forage. There is no evidence to show that animals develop a special craving for the plant.

FIG. 5.—The "timber" milk vetch *(Astragalus campestris)*. Mounted specimen, in nature grows in roundish tufts. *(Photo by Glendenning)*

TIME REQUIRED TO PRODUCE SYMPTOMS

The disease may appear within five days in animals pastured in the affected zone; with sheep as short a time as two days has been reported. In so far as horses are concerned we have not as much information as could be desired; we have however one record of a horse being tethered out where the plant grew profusely, with symptoms developing in about four days.

SUSCEPTIBLE ANIMALS

In so far as cattle and sheep are concerned, by far the most susceptible animals are lactating females, and especially so if strangers to the affected zone. In such class of stock it is possible to find practically all more or less affected, while dry females and males are not showing symptoms: the latter classes may however be affected, and sometimes severely, but as a general rule not so badly as females in milk.

In a very dry season all classes of stock suffer to a greater extent than in an average year. Among range cattle cows with their first calf probably show the greatest percentage of affection. While lactating cows and ewes are the most susceptible, their progeny are not as a rule affected: this, however, is not an age immunity but is dependent upon the amount of the plant they may eat.

Among horses the trouble is not confined so closely to lactating females as in cattle and sheep. The greatest losses occur in animals new to the range. Natural cases have been seen in foals, but in every instance so far as we know these were foals that had lost their dams and were eating the plant. It is possible for calves and lambs to be affected, but this is not likely to occur if they are sucking.

A true immunity does not appear to exist, as an animal may recover only to take the same disease the next summer; in fact it may do so for several successive summers. There is no doubt, however, but that native stock are more resistant than recently imported animals, and that some individuals are able to eat more of the plant without harm than others.

We have no information as to the effect of the plant on other species of animals, except for the following note dealing with a pig: "A sow I had was always digging for the roots of this plant, but always seemed to do well and never suffered any injury from eating milk vetch."

ECONOMIC IMPORTANCE

A number of factors make it difficult to estimate the economic loss due to this disease: recent importation, state of lactation, dryness of the summer, comparative inaccessibility of ranges, and reticence of owners, all play a part. As an instance of the latter we cite a sheep owner who acknowledged a three thousand dollar loss when directly questioned, but who previously had said nothing through fear of injuring the reputation of his range: with cattlemen the chronic types of the disease were thought by some to be tuberculosis.

Although the condition is indigenous to the country it does not appear to have been brought to official attention until 1912, when a sheepman in the Similkameen district suffered heavy losses in sheep recently imported from Montana: it is said that practically all of a band of 900 were affected and that some 400 died (44 per cent). We have other records of a 20 per cent death rate in sheep with 50 per cent of the band affected, but as a rule these figures can be cut in half.

In cattle we have record of 100 per cent affection of lactating animals with a 44 per cent death rate, on the other hand we know of a 30 per cent affection without any deaths. Although native stock are more resistant we know of one herd with 100 per cent affection and death rate of approximately 6 per cent. Ordinarily in a dry year we would consider a 25 per cent affection to be approximately correct for native stock, but it is difficult to estimate correctly; 10 per cent losses are not uncommon and on the other hand may be only 2 or 3 per cent. On the whole the disease is benign in character, but to the death rate must be added, the economic loss due to emaciation and secondary diseases, chiefly pulmonary, and the fact that owing to their weakened condition they are very liable to get into trouble months after they were first poisoned. The loss from these causes probably exceeds that from the actual deaths.

In horses the heaviest loss we know of occurred in a recently imported band of thirty-five head, the mortality being 74 per cent; several other bands are known to have suffered 10 to 40 per cent losses. As a rule, however, the death rate is not high, but secondary pneumonia is liable to occur, and the slightest exertion will bring on an alarming attack of difficult breathing, which often results in death. Some of these cases recover but the majority are never of much use for hard or fast work.

SYMPTOMS

Sheep.—In these animals we have had an opportunity to observe the earliest symptoms, which we have been unable to do in either cattle or horses. These consist of evidence of urinary irritation in some cases (small quantities of urine being passed at frequent intervals), and sudden attacks of heart trouble in which the organ runs wild, as if a governor had been taken off an engine (paroxysmal tachycardia). These attacks as a rule do not last long, but often cause death. Any excitement or unusual exertion is liable to bring them on, and more especially so if the day is hot or the stomach is full of food.

Apart from the urinary and heart symptoms, there are two types of the disease which often occur in the same animal; in the one the gait is mainly affected, in the other the breathing.

Inco-ordination is not confined to the hind legs as much as it is in cattle, the front legs often being affected in sheep. In the early stages inco-ordinate movements come and go. The hind feet may be dragged on the ground and in some cases may interfere with each other. Loss of fetlock control may occur and be extremely well marked in either front or hind legs. Occasionally a string-halty action of the hind legs is noticed accompanied by a stiff gaited action of the front.

Difficult respiration, more noticeable after exertion, is a common symptom, often accompanied by a protruding and cyanotic (bluish) tongue, and by a wheezing or roaring sound. Loss of voice in such cases is quite common. There is usually a cough and occasionally a nasal discharge. Grinding of the teeth has been noticed and a slight bloating, but neither are common. Roaring may be so pronounced as to be heard a good one hundred yards away: this appears to be more often expiratory than inspiratory, which would indicate a bilateral paralysis of the larynx. The appetite is usually good, and consciousness is retained although there may be some indication of intoxication. There is no indication of pain or of fever, except when an attack of dyspnœa is in progress, when the animal struggles to get its breath, during which the temperature may rise one or two degrees. Occasionally there may be a little difficulty in swallowing, and small particles of food getting onto the lungs give rise to a secondary pneumonia. Loss of flesh is a common sequel, but some remain fat.

Cattle.—In general the symptoms are much the same as in sheep. Interferring (knocking) with the hind feet is very common. In some cases a marked dropping of the hind-quarters is seen when a hind leg is advanced in walking. The front legs may occasionally show loss of control, but not to the same extent as in sheep. Sometimes in rising the hind-quarters may be dragged for yards. Loss of voice is common and characteristic, the animal upon attempting to bawl making no sound beyond that of exhaling breath. Difficult respiration, more noticeable after exertion, is common, often accompanied by a protruding tongue as in sheep, and by a wheezing or roaring. There is usually a husky cough, which may persist for months. A greenish coloured discharge from the nostrils is not uncommon and is connected with difficulty in swallowing food properly. Urinary irritation may be in evidence. Excessive salivation when ruminating has been noticed, but is not common. In some the respiratory symptoms are present, in others those of inco-ordination, but usually there is evidence of both. Emaciation is usually well marked, but other animals though visibly affected remain fat. There is no evidence of stupor. Pain is apparently absent and the temperature normal, except as mentioned in sheep. An unusual symptom noted by Dr. George, of Kamloops, was temporary blindness in three cows, which usually passed off in a few hours but in one instance persisted for two days: we have no other record of this being noticed in either cattle, sheep or horses.

Horses.—The early symptoms have not been seen by the writer but from information supplied by ranchers there is some inco-ordination shown. As usually seen the animal appears to be normal when standing undisturbed, but just as soon as it is excited or made to move it shows signs of distress: the breathing becomes laboured; the nostrils fully extended; roaring well marked and of an expiratory type (which is very rare in ordinary roarers), the animal breaks into a profuse perspiration, staggers, falls, may roll, usually defæcates, the mouth wide open emitting foam, and the tongue cyanotic, in fact death from asphyxia appears imminent and may occur. Such a paroxysm lasts from five to fifteen minutes, but has been known to last half an hour. During the attack the temperature rises two or three degrees, but drops back to normal when the animal has recovered. After an attack there is evidence of exhaustion but otherwise the horse appears normal. Very slight exertion will bring on such attacks: we know of one animal that had three when being driven a short distance from pasture, the third proving fatal. One horse is reported as running about 300 yards and then dropping dead with a gush of blood from the nostrils. These animals are usually in fair condition but repeated attacks bring on emaciation, and pneumonia is a common sequel. We are unable to say whether aphonia (loss of voice) occurs as in cattle and sheep, but upon one occasion we heard an affected animal neigh and the voice was certainly affected, being unnaturally hoarse.

COURSE OF THE DISEASE

Death may occur within a few days after the first symptoms are noticed. Such losses are no doubt due to paralysis of the heart. In many cases death may not occur for weeks, and this may be in what appeared to be a mild case: it all depends upon how much strain is put on the weakened heart by excitement or exertion. Some cases die months later from a secondary pneumonia, due to small particles of food getting into the bronchi and setting up an irritation, this being dependent upon the faulty condition of the throat. If animals are brought down to lower levels and away from the plant in good time, they recover slowly, but the voice may be permanently lost, especially in bad cases. Many cattle if fed well through the winter, fatten up during the following summer, if not lactating, and can be beefed, but they often still show some weakness of the hind-quarters and slight interference with the hind fetlocks; such animals can easily get into trouble and die even though beef fat; if they calve the second summer they usually manage to raise the calf but are hard to keep going the second winter. Some animals appear to recover completely, and this can be said for all three species concerned, on the other hand many do not, consequently the economic loss is much greater than would first appear.

AUTOPSY

On autopsy nothing distinctive or outstanding is noticed. In general the lesions are those of cardiac failure or of broncho-pneumonia and anæmia. In some cases small pieces of grass or other food can be found in the bronchi. The heart is usually flabby and the amount of pericardial fluid is increased; pericarditis is fairly common. In some cases the brain and cord appeared slightly congested, in others normal. Microscopically some evidence of degeneration has been found in the vagus nerve and in the superior-lateral columns of the cord, but further work needs to be done with the nervous tissues. Some evidence of nephritis has also been found.

EXPERIMENTS (PRELIMINARY)

In 1915 Hadwen showed that the disease was not infectious by animal inoculation.

In 1916 the writer fed 73 pounds of *Lathyrus ochroleutus* to a sheep with negative results.

In 1917 a supply of *Astragalus campestris* was shipped to Agassiz from Chopaka. During transit this became mouldy. One lamb was fed 114 pounds over a period of 45 days, when it died through getting hung on a wire fence. Upon one or two occasions this animal was thought to be a little uncertain in its gait. Another lamb was fed 37 pounds of the same kind of food: on the thirteenth day was noticed to stand with the hind legs widely separated. On the sixthteen day a similar action was noticed, and when made to move did so slowly and was inclined to drag the hind legs; it appeared to feel the heat. The next day it slipped twice in bringing the hind legs forward and fell, appeared dopey and distinctly weak in the hind-quarters. On the eighteenth day it was weak, had a staggering gait, pulse 54, and unimpaired appetite. Found dead the following morning. On autopsy the only outstanding lesion was pericarditis; slight congestion of the lungs, kidneys and meninges of the brain. Other organs appeared normal.

This case suggested that the plant might be implicated, but was offset by the large amount fed to the other lamb; further at the time we had no information as to the early symptoms seen in poisoned sheep. From what we know now there is no doubt but that the animal was suffering from poisoning, but the plant fed was unnatural, being somewhat dry and moldy, the later shipments, however, were in better shape than the earlier and this may have had some bearing on the negative results in the first lamb.

About this time statements made by various individuals that the plant was good feed directed attention away from it.

In 1921 four sheep native to the range were fed in the mountains near Chopaka at an elevation of 3,000 feet. Each ewe had a lamb at side but the lambs were not sucking at all well and one sheep was found to have the wrong lamb. Feeding period ten days, June 24-July 3. Each sheep (Merino) was placed in a small corral with a lamb; the pens were too small for practical purposes. Pen 1. Fed 70 pounds *A. campestris,* 40 pounds of which was in seed. None of the sheep cared for the plant if at all wilted.

Pen 2. Fed 67 pounds *A. campestris,* 40 pounds in seed.

Pen 3. Fed 72 pounds mixed plants, no Astragalus. Feed consisted largely of *Balsamorhiza sagittata, Rhinanthus crista-galli, Gaillardia aristata, Heuchera ovalifolia, Senecio pseudoaureus, Penstemon confertus, Erigonum heracleoides, Castillejia miniata, Arnica fulgens, Sedum stenopetalum,* species of *Arabis* and other plants.

Pen 4. Fed 19 pounds *A. campestris,* 32 pounds pine grasses (chiefly *Calamagrostis*) and 19 pounds mixed plants.

Results negative, all remained healthy.

Notwithstanding the negative results with sheep native to the range, it was still felt that *A. campestris* was the plant we were looking for, as the sheep were not typical subjects because they were not milking well, nor were their pens large enough to cause them to exercise, and further as we see it now, they were built in the shade. At a later date these suspicions were strengthened by reports of trouble from Tatlayoko lake in the west, and from Windermere lake in the east, both points being outside of the previously known range of the disease. The Windermere lake reports were especially interesting, partly because they came from a veterinarian, but chiefly because they came from a point about 150 miles further east than was known before, and occurred in another climatic belt,

i.e., the Rocky Mountain. From both points the plant was obtained and the information that it was being eaten freely and that it grew in abundance. Further from both places in addition to typical cases in cattle, equally typical cases of the so-called mountain fever of horses were reported, a fact that helped to confirm the suspicion that the horse disease was due to the same cause as the disease we had been working with in cattle and sheep.

DEFINITE A. CAMPESTRIS FEEDING EXPERIMENT

In this experiment four sheep with lambs rather older than was wished, being about four months of age, were shipped from Agassiz to Chopaka. On June 14, 1923, they were uncrated and driven some eight miles to the ranch where we conducted our negative feeding tests in 1921. The weather being unusually bad the sheep were not corralled on the mountain as before, but were kept at an elevation of about 1,200 feet. All the feed was collected at an altitude ranging from approximately 2,400 to 3,500 feet, the vegetation being backward owing to unusually cool weather.

Description of sheep: No. 1, grade Suffolk, 2 years; No. 2, grade Suffolk, 3 years; No. 3, grade Oxford, 15 months; No. 4, grade Oxford, 2 years. Each with lamb about four months old.

Sheep were all placed in one corral, thus allowing more space in which to observe their movements, but having the disadvantage that in event of illness only the approximate amounts of food eaten could be estimated.

Plant fed, *Astragalus campestris,* picked fresh each day and often twice a day. Feeding started the evening of the 14th and was continued to the 26th of June. The plants fed from the 14th to 21st were 20 per cent in bloom; from the 22nd to the 26th, 90 per cent in bloom. Many of the latter had formed pods but in none were the seeds mature. Total amount picked 381 pounds; amount eaten approximately 343 pounds. Results obtained: sheep No. 2, a poor feeder and whose lamb left her to suck No. 3, did not show any symptoms. Lambs No. 1 and No. 2 (lamb numbers correspond with their dam's) did not develop any symptoms, these two lambs got more milk than the others. Positive results in the remaining three sheep and two lambs. The lambs eat as freely as the sheep; in fact at the start slightly better.

Lamb No. 3. This lamb tailed the flock when driven from Chopaka on the morning of the 14th. Got wet during the early morning of the 13th and that night.

On the third day of feeding, June 17, it was noticed at noon to be staggering badly. Approximate amount of astragalus eaten, 7 pounds. Inco-ordinate movements continued during the afternoon and at times it slapped its front feet down in a stiff-legged manner seen in some natural cases. Was considered by the rancher, who had seen hundreds of cases, to be a typical case. Unfortunately this lamb's normal temperature was not known (normal sheep vary tremendously, from 101·3-105·8°, average about 104°), upon examination its temperature was 106, respiration 50 (normal 12-20), pulse too weak and fast to count. At first it was thought the animal had congestion of the lungs. The voice was unnaturally husky but the lamb was not wheezing. Was noticed to urinate and defæcate. Shortly before 6 p.m. it was bothered by a tabanid fly, and in trying to shake itself clear, it lost control of all four legs, rolled completely over on its neck (did not break), and died in a few moments. Autopsy showed some congestion of the lungs, slight congestion of small intestines and fourth stomach, and heart ecchymotic towards the apex.

While it appeared that this animal was suffering from slight congestion of the lungs, subsequent events tended to show that it was also poisoned by *A. campestris,* and that death was due to heart failure. No impaction was found that might have accounted for some uncertainty in gait. The case was con-

sidered typical by the rancher and his opinion was of value as he had lost many animals in previous years. The extraordinary rapidity with which this case developed bore out the statement of sheepmen that cases could develop on the range in two days.

Sheep No. 3.—This animal was in a much better state of lactation than any of the other ewes; after losing her lamb (No. 3) she suckled the lamb of ewe No. 2, who was practically dry.

On the morning of the fifth day of feeding, June 19, was noticed to be staggering. Up to this time she had eaten approximately 14 pounds of astragalus. Still eating and ruminating but looks intoxicated. Groans at times when lying down but does not appear to suffer any pain. Stands wide behind, drags the right front foot more than the left, and knuckles over on the fetlocks. At 8 p.m. was strong enough to bunt a ram lamb that tried to jump her. Temperature, 104·2.

June 20. Very intoxicated. Still ruminating. Temperature, 104·2; pulse 84 and weak; respiration, 28 per minute. Cannot stand properly, goes on knees or knuckles fetlocks, hind legs also bad. Still lets lamb suck her but cannot stand bunting.

June 21. Still eating. Lies down most of the time or moves with the hind legs widely spread and on her knees. Grinds teeth at times. Is beginning to wheeze a little; this wheezing is noticed on expiration. Temperature, 104·4; pulse, 100; respiration, 16 (6.30 a.m.).

June 22. At 6 a.m. temperature 104·5, pulse very weak and 148 per minute, respiration 14. Can move around on the knees. Eats a little. Apparently suffers no pain. Towards evening was seen to walk, but could not do so without spreading the hind legs and knuckling fetlocks. Was noticed at 8.30 p.m. and was resting quietly.

June 23. At 5.30 a.m. was found to have died during the night. It is difficult to estimate the amount of astragalus eaten by this sheep while she was ill, but it is doubtful if she consumed more than 25 pounds from the time feeding started until death occurred; the first symptoms were noticed after she had eaten about 14 pounds.

Autopsy. Lungs hypostatic. Heart ecchymotic and anæmic infarct towards apex. Increased amount of pericardial fluid. Kidneys show some nephritis. Liver slightly congested. Fæces mucous coated. Abomasum congested. Well marked congestion of meninges of brain and cord. Other organs appeared normal.

FIG. 6.—*A. campestris* poisoning. Experimental sheep No. 3. Note position of front legs.

(Original)

FIG. 7.—*A. campestris* poisoning. Experimental sheep No. 3. Note knuckling of front and hind fetlocks. *(Original)*

FIG. 8.—*A. campestris* poisoning. Experimental sheep No. 3. Note position of front feet—the right hind leg is just visible where the the front legs cross. *(Original)*

FIG. 9.—*A. campestris* poisoning. Experimental sheeep No. 3. Unable to stand, but still eating. *(Original)*

FIG. 10.—*A. campestris* poisoning. Experimental sheep No. 3. Note widely spread hind legs, and left front fetlock knuckled to the right. *(Original)*

Sheep No. 4.—On June 19 the fifth day of feeding, towards evening this sheep was noticed to be passing a few drops of urine at frequent intervals. The rancher stated this was often noticed in an affected band, and was undoubtedly an early symptom.

June 20. Same condition noted.

June 21. Was thought to make a mis-step. Was noticed to make a noise like a hiccough twice when ruminating. Still shows evidence of urinary irritation.

June 22 and June 23. Does not appear quite as bright as a normal animal, but shows nothing outstanding.

June 24. After being made to move around the corral for 3 or 4 minutes, she lay down suddenly and nearly died from heart failure; a bucket of water was immediately thrown over her head which revived her so that the head could be held up. Temperature, 103·8; respiration, 50; pulse too weak and fast to catch but was over 200. Five minutes later respiration increased to 76, ten minutes later 48, and thirty minutes after the attack was 44 with a weak pulse of 150. In about an hour she got up but was decidedly groggy.

June 25. Is not staggering but seems dopey. Passing a few drops of urine at frequent intervals. Was noticed to make a mis-step with hind leg, and if made to move fast is beginning to wheeze. Later was noticed to drag the hind feet.

June 26. After five minutes driving around at a walking pace, wheezes slightly, drags hind feet, and urinates frequently. Experiment stopped. If animal had been kept at a higher elevation would probably have died from heart failure.

This sheep was left with the rancher for further observation.

July 4. Wheezing badly.

August 1. Lost her voice and could be heard roaring a hundred yards away.

December 2. Still wheezing but keeps fat, the latter being unusual.

April 14. Wheezed until Christmas, then got better and had a fine lamb.

Lamb No. 4.—June 19 the fifth day of feeding, was thought to be dragging right hind foot.

June 20th. Was noticed to be dragging the left hind foot several times and the right hind once; appeared slightly groggy.

June 21. Lamb is indoubtedly diseased. Drags the hind feet and occasionally the front fetlocks knuckle over. Voice not affected, still sucking and eating the astragalus.

June 22. Is still quite strong on feet, although unsteady. There is a suspicion of wheezing.

June 23. After being run for a few minutes breathes with the month open. Voice not affected.

June 24. Seems stronger if anything.

June 25. Breathing noisier than before; towards evening wheezing quite pronounced and is beginning to lose control of the voice.

June 26. After driving around for a few minutes is wheezing badly, inco-ordination well marked, knocks the hind feet together like an affected cow, voice unnatural, urinates frequently. Upon further driving breathing is very much worse, the tongue being protruded. As a check was wanted on sheep No. 3 and the time of feeding was concluded, the lamb was killed. Autopsy was practically identical with that of the sheep in question. Kidneys congested, heart ecchymotic and anæmic infarct near apex, meninges of brain and cord congested, lungs and other organs appeared normal.

FIG. 11.—*A. campestris* poisoning. Experimental sheep No. 4 and lamb No. 4. The lamb has its mouth open. Both developed typical cases of the wheeezing or roaring type of the disease. *(Original)*

Sheep No. 1.—On June 23 after moving around was wheezing slightly. This was also noticed when the animal was eating, but did not last for more than fifteen minutes. June 25. Was wheezing a little. June 26. Wheezing slightly after walking around for a few minutes, and is also beginning to pass a few drops of urine at frequent intervals. No signs of inco-ordination. Experiment stopped.

To sum up—the feeding of *Astragalus campestris* produced nervous symptoms typical of natural cases in three out of four sheep and two out of four lambs. As occurs in nature, the symptoms were not identical in each case, some showing the inco-ordinate and others the laryngeal type of the disease. It is quite evident that the heart is severely affected, and that even more pronounced results might have been obtained if feeding had been conducted at a greater altitude. The fact that the heart is affected explains why animals with full stomachs, or during hot weather, suffer more, and also why excitement or exertion aggravate the symptoms. The beautiful case in sheep No. 3 and the equally typical case of another type in sheep No. 4, as compared with the light symptoms in sheep No. 1 and the negative results in sheep No. 2, may be largely attributable

to the state of lactation. Sheep No. 2 was practically dry, her lamb being taken by sheep No. 3. The cases in lambs are interesting because it is very rare in nature to see such animals affected, under natural range conditions, however, they would not have to eat as much of the plant as the experimental lambs were compelled to do.

One of the affected animals was showing symptoms on the third day of feeding, and three others were showing signs of poisoning on the fifth day, thus supporting the field evidence that cases may occur in less than a week.

TREATMENT AND PREVENTION

Affected animals should be brought down from the incriminated zone to as low a level as possible. If this is impossible they can be moved higher up the mountain where the plant does not occur; this, however, is not advised if sufficient feed can be found on the lower levels, as increased altitude may aggravate the symptoms. Such animals should not be excited or hurried, and special care must be exercised if the day is very hot. The difficulty of treating range animals is fully realized, but will be outlined, as notwithstanding its impracticability in some cases it may be of use in others.

In acute cases that suddenly drop and which may die of heart failure, a bucket of cold water dashed over the head may cause them to revive. If able to swallow, a stimulant such as aromatic spirits of ammonia would be useful; this should be given as a drench, and with care because the throat may be partially paralyzed, the dosage being as follows: horses and cattle, from 1 to 2 ounces, sheep 2 to 4 drams, to be given well diluted with water. After an attack a few small doses of tincture of digitalis may be useful to improve the tone of the heart, but its use is limited because in these cases the trouble lies more with the nerves governing the heart, than with the organ itself: the dose of tincture of digitalis for horses and cattle is 2 to 4 drams, sheep $\frac{1}{2}$ dram to $1\frac{1}{2}$ drams, given with sufficient water to make a drench, and usually given 12 hours apart, but in acute cases may be given in small doses for a couple of times about two hours apart. The only hopeful treatment lies in removing the animals from the plant, and putting them on a course of nerve tonics; if this is done in reasonable time many will recover and even chronic cases are benefited. This consists of the following: powdered nux vomica in doses of 1-2 drams for horses or cattle, 20-40 grains for sheep (480 grains to the ounce). It is recommended that the smaller doses be tried first, that they be given at least once, and for preference twice, a day for two weeks or if necessary for a month. The chief difficulty lies in giving the drug to range animals. Ordinarily it is given thrown back on the tongue with a spoon, or with feed; in the latter, however, it is often refused on account of its bitterness. One has also to recollect that more harm than good may be done if some of these cases are handled roughly. It can be managed however with many horses, gentle cows, and sheep. Efforts to feed the drug mixed with salt have been tried but up to the present without success, and if such a method of mixing with other materials can be devised one has always to guard against any one animal getting too much of the drug. Its main constituent is strychnine which gradually accumulates in the system, its use, therefore, should be discontinued for a week if an animal shows signs of excitement, restlessness, or twitching of the muscles.

It is possible that the difficulty with range cattle could be overcome to a great extent by following the system adopted by Marsh in dealing with locoed cattle: this consists of driving the animals into a chute, and then injecting them with a hypodermic syringe. Injection should be made under the skin, back of the shoulder being a good place, and if strychnine sulphate is used in one half grain doses for cattle or horses, it should be used once a day for ten days, then stopped for two or three days, and again continued if necessary. Animals should be watched for any of the symptoms mentioned above, and if they occur treat-

ment must stop at once. No trouble should arise with this dosage of strychnine as it is smaller than that contained in the nux vomica dose, which has been reported upon by ranchers as giving good results: there is however always the chance of some individual animal being peculiarly susceptible to its action.

The extraordinary rapidity with which the "timber" milk vetch *Astragalus campestris* can produce lesions of the nerves that, so far as the writer knows, are only comparable to those seen in chronic lead poisoning or lathyrism, does not indicate the possibility that any antidote is likely to be found for its poison. Lathyrism and locoism, two diseases caused by members of the pea family, both remain without an antidote although they have been known for many years.

PREVENTION

In different sections different causes (and their number is legion), have been advanced as being responsible for the disease concerned. It has however been shown that such a disease is always associated with the "timber" milk vetch, *Astragalus campestris*, and that typical symptoms follow when it is fed experimentally. Prevention therefore lies in limiting the consumption of this plant by animals.

To eliminate the plant from the ranges would appear to be out of the question, as it is firmly established.

Heavy losses are intimately connected with shortage of other food, and ultimate success must lie in a correct distribution of stock over the spring, summer, and fall ranges to prevent overgrazing.

To prevent the occurrence of the disease, lactating cows or ewes should not be ranged in the Arid Transition zone (which runs from approximately 2,000 to 4,000 feet), from the latter part of May until towards the end of August. The last half of June and July are especially dangerous. Such animals can be kept either above or below the zone with safety, in so far as this disease is concerned. Dry cows and ewes and male animals may be run in the affected zone, except in a very dry year, as although such animals may be affected and sometimes severely, they are not so susceptible as lactating females. Horses of all kinds, unless native to the range, should not be pastured in the affected zone.

Affected animals should be removed from the zone upon showing the slightest symptoms, and for preference to lower levels, but if this is impracticable they can be taken higher up the mountain. Such animals must not be hurried or excited.

SUMMARY

(1) *Astragalus campestris* Gray is added to the list of poisonous plants.
(2) For descriptive purposes in British Columbia we suggest that the plant be called "the timber milk vetch".
(3) Its habitat is the Arid Transition zone which lies between 1,600 and 4,200 feet approximately.
(4) It causes heavy losses in horses, cattle, and sheep.
(5) In cattle and sheep lactating females are especially susceptible, but dry females and male animals may be affected.
(6) Poisoning takes place from the end of May until the latter part of August; most cases occur in June and July.
(7) It affects the nervous system.
(8) The disease produced is new to science, and it is suggested that it be called " pseudo-lathyrism".
(9) Of its many local names that of "timber paralysis" is favoured, with "roaring disease" as second choice.
(10) Symptoms may develop in less than a week after being exposed to the plant.
(11) Two types of the disease occur, one characterized by inco-ordination, the other by difficult respiration: in many cases they are combined.

(12) In cattle and sheep the disease is characterized by loss of voice, lack of leg control, and roaring.
(13) In horses by roaring (bilateral laryngeal paralysis, also in cattle and sheep), alarming attacks of difficult respiration, that occur upon the slightest exertion, and in the early stages inco-ordination.
(14) Sudden death from heart failure often follows excitement or exertion, the inhibitory power of the vagus being lost (paroxysmal tachycardia).
(15) Recovery may take place, but chronic cases last for months: emaciation and pneumonia are common sequels.
(16) The use of nux vomica has proved beneficial.
(17) Both types of the disease have been produced experimentally in sheep.

FIG. 12.—*A. campestris* poisoning. Natural case of the roaring type with loss of voice. Note the protruding tongue.
(Photo by Hadwen)

FIG. 13.—Natural case, *A. campestris* poisoning. Note the tongue and emaciated condition. Such an animal would show inco-ordination of hind legs and loss of voice. The calf is sucking and is not affected.
(Photo by Hadwen)

FIG. 14.—*A. campestris* poisoning. A common type of case that shows emaciation, and would hook the hind feet when walking.
(Photo by Hadwen)

FIG. 15.—*A. campestris* poisoning. Range animal in fair condition but suffering from heart attack after excitement of being roped.
(Photo by Hadwen)

FIG. 16.—*A. campestris* poisoning. Evidently the same animal as Fig. 15, beginning to recover from attack. *(Photo by Hac'wen)*

FIG. 17.—Typical range of the Douglas fir type at 3,500 feet. The speckled appearance of the foreground is due to *Astragalus campestris* in bloom. *(Original)*

LUPINES (*Lupinus* sp.) (Fig. 18)

The lupines, sometimes known as blue peas, blue beans, horse beans, etc., are widely distributed in the province, and twenty-one species are recorded of which at least four are reputed to be poisonous (*L. sericeus, L. leucophyllus, L. argenteus* and its variety *argophyllus*). The lupines are not easily differentiated even by one who has a little botanical knowledge, and for the purposes of this paper will be treated as one plant.

FIG. 18.—Lupine.
(From painting by Young—photo by Glendenning)

Notwithstanding their number and their wide distribution, they are not of much importance as poisonous plants in this province. While other species of animals may be affected, the majority of cases occur in sheep: our records would indicate that an occasional horse is affected and sometimes sheep.

As with a number of other plants it is the hungry animal that is poisoned. Cases rarely occur in a band of sheep if they are well fed. The following extract from a letter can be used to illustrate how poisoning occurs: "Yearly, when I take the flock to summer pasture I lose one or more by lupine . . . after the first day however they seem to eat it abundantly and show no ill effects". The point here is, that unless the band is held and allowed to fill-up, they come upon the lupines, hungry, which being more easily reached than the grasses are eaten in greater quantity over a short space of time, than occurs later. Poisoning does not occur except when a certain amount is eaten more or less at one time, because the poison does not accumulate in the system but is quickly passed off, chiefly with the urine. This fact explains why sheep regularly pastured in lupine country, can eat over the course of a day far more of the plant than would poison them than if taken in, say, one hour. In taking sheep across a lupine patch it is much safer to let them drift across than to drive them.

The time that lupines are most dangerous is when they are in pod. Marsh has shown that the seeds are especially poisonous, and that it only requires approximately one and a half pounds of the fully developed pods with seed to kill a 100-pound sheep.

SYMPTOMS IN HORSES

Muscular twitching, constipation, dullness, and a tendency to lift the front feet higher than usual.

SYMPTOMS IN SHEEP

These may show in one or two hours after feeding on lupine, but are sometimes delayed for nearly a day. Death may be very sudden, or not until two or three days.

Drooping of the ears is commonly seen in the early stages. Quite often excitement is shown, the animals butting into other animals or objects. In some cases the head is kept pushed against something solid.

In mild cases the breathing is heavy and laboured, the animal passes into a condition resembling sleep, and may snore. In more acute cases, the breathing is extremely difficult, the animal struggling convulsively to get its breath; death may occur during one of these attacks, the animal dying in convulsions. In some cases the animal lies in a completely insensible condition until death occurs without a struggle.

TREATMENT

No medicinal treatment of any value is known for lupine poisoning. Most of the trouble will be avoided if care is taken to keep hungry sheep from patches of lupine that are in pod.

THE PARSLEY FAMILY *(Umbelliferae)* (Figs. 19 to 25)

Under the Umbelliferae we have to consider the poison-parsnip or water-hemlock of which there are at least two kinds in the province, the common one on the coast being *Cicuta Douglasii* and that of the interior *C. occidentalis.* The main difference between the two is in the rootstock. Both are equally dangerous and are considered the most violently toxic of all plants growing in the province. As the common name water-hemlock would imply the plant grows in wet places, and in such places other umbelliferous plants grow which may be confused with it. The plant that seems to be the most often mistaken for water-hemlock is the water-parsley (*Oenanthe sarmentosa)* which grows abundantly in wet ground near the coast. Another water loving plant that may be confused is the water-parsnip (*Sium cicutaefolium*). Angelica may also be mistaken for cicuta but it does not grow in such wet ground; on one occasion we fed three pounds of *A. genuflexa* to a calf with no ill results, thus confirming the generally accepted opinion that it is harmless. *Oenanthe sarmentosa* has never caused trouble. *Sium cicutaefolium* is reported by some writers as being poisonous, but so far as I can find, only on circumstantial evidence. To settle the point in so far as the plants growing in British Columbia were concerned, the writer watched nine cattle (four cows, three two-year-old, two yearlings) turned into a field and overnight found that they had eaten 138 plants without ill effects. Further a calf was fed in two days 19 pounds of the plant in flower including the roots, and a sheep was fed 6 pounds, without either animal showing any symptoms whatever.

Two other poisonous umbelliferous plants remain to be mentioned—*Conium maculatum* and *Berula erecta.* The first mentioned is the poison hemlock, an erect, smooth, branching plant, from 2 to 6 feet high, with spotted stems, and large decompound leaves, which occurs only in the vicinity of Victoria where it is not likely to trouble live stock. This plant, famous from ancient days as

extremely poisonous, has a disagreeable odour and is seldom eaten by stock; it had a mousey smell especially noticeable when the plant is bruised. It is said to have caused poisoning in the human in several ways—the seeds have been mistaken for aniseed, the leaves for parsley, and the roots for parsnips; also from blowing whistles made from the hollow stems. *Berula erecta,* the creeping cut-leafed water-parsnip whose range is given by Henry's Flora as streams and ponds, Kamloops; Okanagan Valley, and eastward, was upon one occasion held responsible for trouble in cattle at Popkum, which is in the lower Fraser Valley and entirely outside of the range mentioned.

Fig. 19.—The water-hemlock *(Cicuta Douglasii).*
(Photo by Glendenning)

HOW TO DISTINGUISH THE WATER-HEMLOCK *(Cicuta)*

In the first place it is a water-loving plant, and therefore should not be confused with the poison hemlock (*Conium*) or with angelica. Of the other water-loving plants we have mentioned, i.e., the water-parsley (*Oenanthe*), water-parsnip (*Sium*), and creeping cut-leafed water-parsnip (*Berula*), the only one that resembles *Cicuta* in stoutness and height is *Sium.* Either plant grows from 2 to 5 feet in height, the stem of *Cicuta* is often purplish but not always, and there is an entirely different arrangement of the leaves as can readily be seen by referring to the photographs. The stems of *Oenanthe* are weak and reclining, from two to three and a half feet long; *Berula* averages from six inches to two feet in height and its stems are weak compared to either *Sium* or *Cicuta.*

Probably the best way for the non-botanist to distinguish the water-hemlock is to examine the root, which in a well grown plant is quite large. If the rootstock (just below where the stem starts) is cut lengthwise, it will be noticed that it has a number of transverse chambers. These chambers are not always as distinct as shown in the illustration, but they are always present, and it is by them that the plant can be readily distinguished from most plants growing in similar situations. The chambers are not so distinct in the spring as they are later in the season.

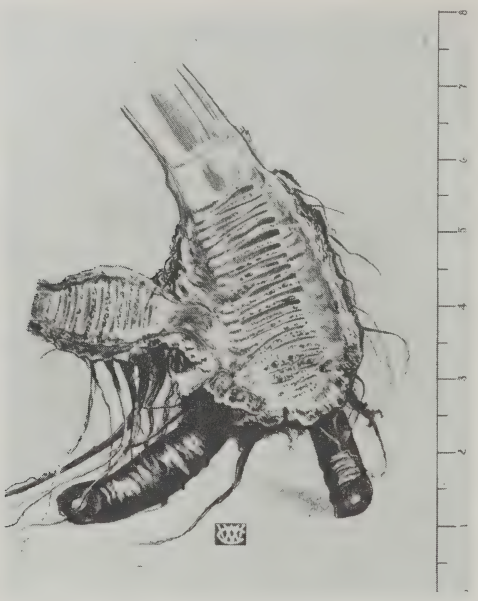

FIG. 20.—Water-hemlock (coast form) showing transverse chambers of the rootstock.
(From painting by Young—photo by Glendenning)

POISONING BY WATER HEMLOCK

Cicuta poisoning is relatively common and, so far as we know, may occur in all kinds of animals as well as in human beings. The majority of cases occur in cattle. Until comparatively recent times all parts of the plant were considered dangerous, but experimental work by the United States Department of Agriculture has shown that the upper parts of the plant as found in hay, never occasion losses. Cases of poisoning may occur in the early spring through eating the young shoots, but it is probable that in doing so a small piece of the root is often taken. The rootstock and roots are violently poisonous, and although they may vary a little in toxicity according to season, a very small amount is sufficient to cause death.

SYMPTOMS AND TREATMENT OF CICUTA POISONING

In some cases death takes place in less than an hour and consequently the first indication of trouble is often the finding of a dead animal. In early cases usually the first symptom seen is frothing at the mouth, followed by uneasiness and pain. This is succeeded by violent intermittent convulsions, in which the animal kicks, sometimes extending the legs rigidly, throwing the head backwards,

bellowing and groaning as if in intense pain. Spasmodic contractions of the diaphragm occur as if the animal were trying to vomit. The pupils are dilated and the eyes sometimes turned in or down. In fatal cases the convulsions become more violent until ended in death.

TREATMENT

No effective remedy is known and in severe cases the convulsions are so violent it is impossible to give anything by the mouth. Cases sometimes occur in human beings, the roots being mistaken for parsnips, artichokes or horseradish, and in such cases if an emetic is given promptly so that the stomach is emptied quickly, recovery usually takes place. Because of the fact that cattle cannot empty the stomach by vomiting, this treatment cannot be applied to them. Chloral hydrate, opium and morphine have been used to control the convulsions, and large quantities of melted lard, linseed oil and milk have been given. Occasionally an animal recovers but when it does, it is probably because the amount of poison taken was very small and not because of any treatment it received.

PREVENTION

The plants can be fenced off, or pulled up and allowed to dry out of the reach of stock, and then be burnt. As they seldom occur in great quantities and are easily dug up, the cost of clearing the average farm of this dangerous weed is slight.

FIG. 21.—Young plant of water-hemlock *(Cicuta sp.)*
(Photo by Glendenning)

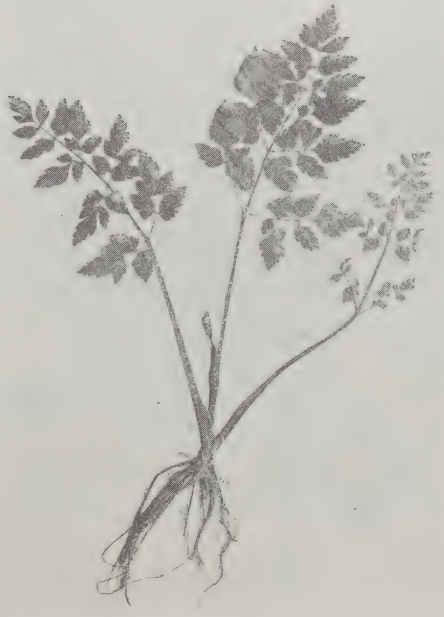

FIG. 22.—Young plant of the water-parsley *(Oenanthe sarmentosa)*. Not poisonous but often confused with cicuta.

(Photo by Glendenning)

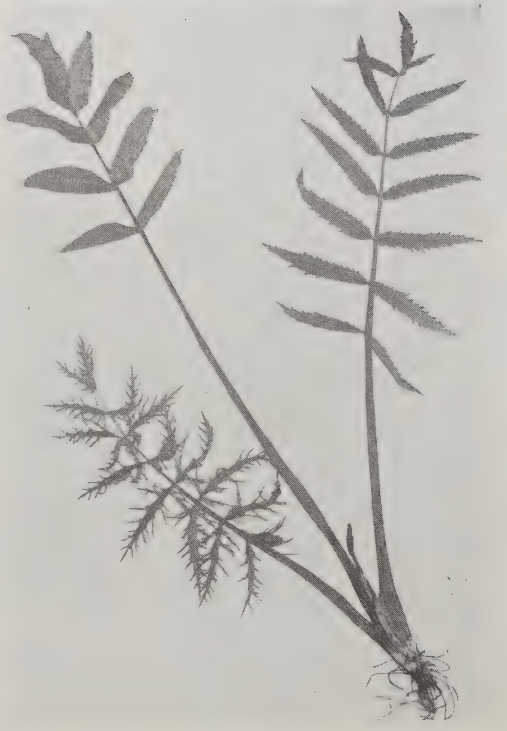

FIG. 23.—Young plant of the water-parsnip *(Sium cicutaefolium)*. Not poisonous in British Columbia, but often confused with cicuta.

(Photo by Glendenning)

FIG. 24.—Young plant of angelica *(Angelica genuflexa)*. Not poisonous, but occasionally confused with cicuta.

(Photo by Glendenning)

FIG. 25.—The cut-leafed water-parsnip *(Berula erecta)*. Poisonous, but not widely distributed.

(Photo by Glendenning)

BERULA ERECTA *(Cut-leaved water-parsnip)*

This plant which is known to be poisonous has not, so far as we are aware, been reported as causing trouble heretofore in Canada. Although our evidence is only circumstantial, it is strong, and is submitted as being of possible interest.

The point at which trouble occurred is approximately 80 miles from the coast in the lower Fraser Valley and outside of the hitherto recorded range of the plant.

The poisoning we refer to took place on July 20 at which time many of the plants were less than six inches in height. A small field that had not previously been used for cattle, had twelve animals turned into it: on the first day two were taken ill, and as soon as this was noticed all were removed. A small stream running through the farm was found at this point to contain a large number of the plants, whereas in another field where the cattle had been previously grazing without harm, none of the plants occurred. Of all the plants in the field or in the stream, none were found that might account for the symptoms except the cut-leaved water-parsnip, and as there was evidence that it had been eaten and no further trouble developed after the animals were moved, there is but little doubt that it was the incriminating agent.

The symptoms as described by the owner were as follows: The cattle were ill for six days. The first two days they were badly bloated but on the first day were given baking soda, punctured with a trocar and canula and well kneeded until relieved and given two pounds of epsom salts. The jaws were set so tightly they had to be pried open. The eyes were staring (pupils probably dilated). Breathing difficult. Badly constipated and the dung covered with mucous. The appetite was lost and there appeared to be some difficulty in swallowing. Stiffening of the legs was noticed. These two animals, cows, giving three gallons of milk per day, went completely dry for twenty-four hours. They laid down practically all the time for six days. Eventually recovered.

None of the following symptoms were noticed: Frothing at the mouth, colic, convulsions, bellowing, blood in urine or dung, rolling of the eyeballs, twisting of the neck or reeling in circles.

ACKNOWLEDGMENTS

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